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ALCOHOLISM AND PULMONARY CONSUMPTION.

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Any one who studiously watches the evolution and dissolution of families, some of whose members are addicted to alcoholic excess, must be struck with the frequent occurence of pulmonary phthisis among them. So on the other hand, it is no less astonishing to find the latter disease suddenly appearing in families who are absolutely free from a phthisical history, and who seemingly live amidst the most healthful surroundings. Why these two conditions should be so closely associated, if in consonance with the current belief, the one is a nervous, and the other a strictly pulmonary disease, is not very clear. The following pages shall be devoted to an elucidation of this intricate problem, in which I shall endeavor to show that these two apparently isolated phenomena are naturally interchangeable with each other, and that like two diversified islands cropping out above the surface of the ocean without exposing their connection beneath, they find their common bond of union in a disordered state of the nervous system.

In order to make this subject as practical and as intelligible as possible, I shall at the very outset endeavor to prove the intimate association between alcoholism and phthisis, how one link may change place with the other in the chain of vital persistence, by citing a number of living, illustrative examples. The first ten of these cases have been culled from the extensive experience of the editor of this JOURNAL, and have been placed at my disposal through his kindness; while the remainder have been obtained from various other sources.

Case I. J. B., aged 42 years, began the excessive use of spirits after the death of his wife. He was a merchant, temperate, prosperous, and a man of character. He became a steady drinker, and was practically intoxicated all the time. After an attack of delirium tremens he was placed under my care. During the four months while under treatment, he was alternately depressed and elated. He complained of wandering pains, and changeable appetite, as well as of spasmodic periods of coughing. A few months after he left me, he relapsed and continued to drink until he died a year later.

His mother and two sisters died of consumption. His father died from injury, but his grandfather was asthmatic, and used spirits to excess for years. One uncle on his father's side died from excess of drink, and another one died of consumption. One uncle died from phthisis after many years of drink excess.

His grandfather on his mother's side drank more or less all his life, and died from some rheumatic trouble.

Case II. B. A., aged 35, a mechanic, began to use spirits for insomnia and general debility, and finally became a periodical inebriate. He was under treatment for six months, and recovered. His father, grandfather, and two uncles, died of consumption. His mother was hysterical, and his grandmother on his mother's side died of some lung trouble. One brother died from chronic alcoholism, and a sister is a drug-taker.

Case III. C. H., age 48, an army officer, began to drink during the late war. He is now a dipsomaniac, with distinct free intervals of three months. His mother died of consumption two months after his birth, and his two sisters died of the same disease. His father's family is temperate, but several members have died of consumption. His grandfather on his mother's side was a sailor, and drank to excess at times.

Case IV. D. P., age 38, a farmer. His drinking seems to date from a nervous shock following the burning of a barn by lightning. His two brothers are chronic inebriates, one

sister is a morphine taker, and the other uses both spirits and drugs to excess for all kinds of imaginary evils. On his mother's side, a grandfather and three aunts and one uncle died of consumption. His mother is still living. His father died of pneumonia, and his grandmother on his father's side died of consumption.

Case V. E. J., age 31, a clerk of inferior mental and physical development, began to drink at puberty. Consumption has been the common family disease of both parents. On his mother's side both consumption and inebriety have been common. On his father's side consumption alone has prevailed.

Case VI. P. O., age 28, is without business, and drank from infancy. He is now a chronic inebriate and has had delirium tremens. His father and two uncles died of consumption. His mother is a woman of wealth and fashion, and she lost her mother and one sister from consumption.

Case VII. M. B., a lawyer, 54 years old, who began to drink at fifty from no apparent cause. His father and grandfather died of consumption, at fifty years of age.

Case VIII. D. T., age 38, a conductor, began to drink after an injury to the spine. A brother, who was injured at the same time, died of consumption. The mother and a sister, the grandfather, and grandmother, on his father's side, all died of consumption.

Case IX. D. B., 24 years old, and without business, began to drink at puberty, and is now a chronic inebriate. Both parents died of consumption. His grandfather on his father's side, and two uncles on his mother's side, died of the same disease.

Case X. A. H., 34 years old, a physician, took morphia for malarial poisoning, and then used alcohol to great excess. His mother and three uncles on his father's side died of consumption. His older brother became an inebriate at about 30 years of age, and one sister is in Colorado to prevent consumption.

Case XI. (*Quarterly Journal of Inebriety*, Oct., 1888, p.

390) " George Ulmer came from England in 1798 and settled at New Haven, Conn. He was a harness-maker, a beer-drinker, and after middle life drank rum to excess, until death at sixty-one years of age. His wife was a healthy woman, and lived to eighty years of age. Eight sons grew to manhood and married. Six of them died of consumption under forty-five years of age. One was killed by an accident, and one died from excessive use of spirits. Two daughters grew up and married ; one died of consumption, the other in childbirth. They left four children; two were inebriates, and the others were eccentric and died of consumption. Of the children of the eight sons only ten grew up to manhood. Four of these drank to excess and died. Three of the six remaining died of consumption, and two others were nervous invalids, until death in middle life. The last one, a physician of eminence, has become an inebriate and is under care at present. He is the only surviving member of all this family. The male members of this family were farmers, tradesmen, and men of more than average vigor in appearance. They married women (so far as can be ascertained) without any special hereditary history of consumption or inebriety."

Case XII. (*Ueber die Trunksucht und ihre Erblichkeit, von* Dr. J. Thomsen, Archiv. f. Psychiatrie u. Nervenkrankheiten. Band 17, 1886. Seite 536) abstract: Father was an inebriate until after he was forty years old, at which time a cardiac affection developed itself from which he ultimately died, but which had the power of restraining him from exercising his morbid appetite. His brother was a drunkard too. Three of his sons became confirmed alcoholics, one daughter died of phthisis, and another son died of general paralysis.

Case XIII. Dr. Grasset. (*Scrofulous and the Tubercular Diathesis*, Brain, vol. 7, p. 19) condensed: Father violent, an alcoholic, and a libertine. Mother is very nervous, and died of cancer of the uterus. Many of patient's relations are drunkards. Her brother and sister died of chest disease, and another brother is always ill, and coughs a great deal. She was admitted May 3, 1879. One month previously she had a chill, rigors, and feverishness, which confined her to bed

for four days; then she began to cough, and had two copious hæmoptyses. She sweats profusely at night, is losing flesh, and in a word has all the symptoms of pulmonary phthisis. Physical examination shows evidence of tuberculosis of both apices.

The histories of these cases give the most unmistakable proof that alcoholism and phthisis are not mere coincidences, but that they have a relationship so intimate that one may be converted into the other. The problem arises, however, as to the channel through which alcohol produces phthisis; for if these two conditions are interchangeable, it is obvious that they must possess a common physiological basis, and this I believe resides in the nervous system. I have elsewhere* (to which I beg to refer the reader) given good reason for believing that pulmonary phthisis is principally nervous in character, and by considering it as such, the natural association between the two diseases is at once established. For whatever else may be said of the action of alcohol, it is pretty generally understood that it possesses a special affinity for the nervous system, and that it produces its principle ravages in the body by operating on this, and by preference on the peripheral nervous tissue. Dr. James Jackson, in this country, and Dr. Wilks, in England, were, I believe, the first to point out this form of disease, and they called it alcoholic paralysis. It has since then received the more appropriate name of alcoholic neuritis, and it is characterized in its early stages by numbness, tingling, hyperæsthesia in the extremities, and later on by anæsthesia, paralysis of motion, loss of knee jerk, quickened pulse, shortness of breath, and frequently by pulmonary embarrassment. The brain and spinal cord remain comparatively normal. The morbid changes occurring in the peripheral nerves under the influence of alcohol are parenchymatous and interstitial, or in other words they are confined to the nerve substance itself, or to its investing membrane. As a rule these changes

* Pulmonary Consumption considered as a neurosis. *Therapeutic Gazette*, Nov. and Dec., 1888.

occur together, the latter in many instances depending on the former, but frequently one exists exclusively of the other; especially is this true of the degeneration of the nerve fibre itself.

It being established, then, that alcohol has the power of producing degeneration of the nerve fibres, it does not require a reckless flight of fancy to see how, by operating on the same tissue, it may bring about that peculiar destruction of lung substance known as pulmonary consumption. Degeneration of a nerve implies degeneration of the organ which it supplies with sensation and motion. Thus, degeneration of the sciatic nerve is followed by impairment of sensation and motion in the muscles and other textures of the leg — a condition which is almost constantly present in chronic alcoholism, and degeneration of the pneumogastric nerves is just as naturally followed by disease of the lungs, heart, stomach, and all the other organs supplied by them. This is no more than we may legitimately anticipate; for it has been amply proven that division of, and protracted pressure of tumors, aneurisms, etc., on the pneumogastric nerves are capable of calling forth all the destructive lesions of pulmonary phthisis.

The following cases will serve to illustrate the close anatomical and physiological association of chronic alcoholism and phthisis, as well as other destructive pulmonary changes with degeneration of the vagii, and of the respiratory center (the latter of which practically amounts to the same thing), and with that of the peripheral nerves. The difficulty encountered in this research has not been so much in obtaining an abundance of material in which phthisis was evidently the direct result of alcoholic abuse, as it has been in finding the records of cases possessing all the points which I desire to emphasize in this paper, viz.: the coexistence of pulmonary disintegration, alcoholism, and nerve degeneration, well brought out by a thorough *post mortem* demonstration.

Case XIV. Drs. Oppenheim and Siemerling (*Archiv. f. Psychiatrie und Nervenkrankheiten*, Bd. 18, S. 507), male, addicted to alcoholic excess, was received in hospital Jan. 26, 1886. He was weak and stiff, but had no pain. At the end

of the same month he became delirious, and also paretic in both legs and arms. Death occurred in March of the same year. On section it was shown that the heart was normal, and that he had pneumonia; microscopically it was proven that the radial, peroneus, and saphenous nerve had undergone degeneration. Not stated whether the vagii were examined or not.

Case XV. Drs. Oppenheim and Siemerling (*Ibid*, p 506). A female, age 45 years, suffering from chronic alcoholism, was received Dec. 26th, and died on the 28th of the same month, in the year 1885. On section there was found chronic exadative pleurisy on right side, as well as a caseous broncho-pneumonia and tracheitis. The great saphenous and superficial peroneus nerves had undergone parenchymatous degeneration of a medium degree. No other nerves were examined.

Case XVI. Dr. T. Déjerine, (*Deutsche Med. Zeitung*, 1887, p. 711.) Female, age 46, a hard drinker, suffered from paralysis of both upper and lower extremities. Had a pulse of 150–160, and her heart sounds were normal. Her death was caused by pneumonia. Section showed parenchymatous neuritis of the cutaneous and muscular nerves, as well as of both vagii in the cervical region.

Case XVII. Prof. Schultze (*Virchow, Archiv.*, Bd. cviii, Heft 2, *Neurologisches Centralblatt*, Bd. vi, 1887, S. 271). Male, 39 years old, developed diabetes insipidus in 1882, but had been feeble since childhood. He used alcohol greatly to excess in his younger days. Some time after the year 1882, he began to suffer from nystagmus, trembling in the arms, perversion of sensation (paraesthesia) in the legs, and from thoracic constriction. In 1886 he became subject to marked attacks of dyspnoea, and death was caused by paralysis of respiration. Section: Degeneration of medulla oblongata and spinal cord, as well as that of the root of the vagus and hypoglossus. No account of the *post mortem* appearances of the lungs is given, but it is evident that these organs were implicated in the morbid processes, since death was produced through pulmonary paresis.

Case XVIII. Strümpbell, (*Archiv. für Psychiatrie u. Nervenk.* Bd. 14, S. 339). Male, aged 47 years, a potator, was received Nov. 25, 1881. His frame is large and powerful. Both of his arms hang helplessly by his side; hands œdematous, skin and tendon reflexes wanting; legs weak and powerless; pulse, 124; temp. 38.2°; deglutition and power of speech impaired; after a while œdema of lower extremities, cough, diarrhoea, dyspnoea; bronchial râles, paralysis of diaphragm, and death, Feb. 13, 1882. Section: Marked tubercular phthisis of both lungs. The radial median, crural, and sciatic nerves were degenerated very decidedly, and Dr. Strümpbell believes that the phrenic and vagii were also involved, but he failed to examine them closely.

Case XIX. Drs. Oppenheim and Siemerling (*Ibid*, Bd. 18, S. 114). Male, 26 years old, a potator, was received in the Charity hospital Jan. 17, 1881, on account of delirium tremens. He complained of headache, giddiness, and formication in the legs. He improved and was dismissed, but was received again on July 28, 1883, on account of marked disturbances in the nervous system. He now suffered from complete impotence, lancinating pains and rectal tenesmus. In August, he became subject to polydipsia and polyuria; on the 12th of December, there was dullness in left supra clavicular fossa, and infiltration of both apices and tubercle bacilli were found in the sputum. He gradually sank and died in August. Microscopic examination showed degeneration of the medulla oblongata, and of all the peripheral nerves which were examined.

Case XX. Dr. Oswald Vierordt (*Neurologisches Centralblatt*, Bd. v, S. 421, 1886). Male, 30 years old, much addicted to alcohol, and without a syphilitic history, suffered since March, 1884, with piercing, lightning pains in the lower extremities, as well as with weakness, unsteadiness, and formication in the same. He also developed tubercular phthisis and died the following March. Section: extended tuberculosis of the lungs and degeneration of the columns of Goll, medulla oblongata, and the cervical and dorsal portions of the spinal cord.

Case XXI. Mr. Sharkey (*British Medical Journal*, 1888, April 21, and JOURNAL OF INEBRIETY, Jan., 1889, p. 67) related a case of alcoholic paralysis of the phrenic, pneumogastric and other peripheral nerves before, and presented specimens of the same to the Pathological Society of London. The patient was a female and addicted to the excessive use of alcohol. She suffered from weakness in her legs, numbness and cramps, and was incoherent in speech. Respiratory sounds were harsh, and in a few days after admission had a rigor, which was followed by a temperature of 102.8°, severe attacks of dyspnoea, paralysis of the diaphragm, and difficulty in swallowing. Respiration 40 per minute, and average pulse rate 140. Spitting of blood supervened, the lung apices began to break down, and she died after having been under observation nearly one month. Section: tuberculosis of both apices and inflammatory changes in the phrenic, pneumogastric, and popliteal nerves.

In these examples we have proof that pulmonary phthisis can be produced through the toxic action of alcohol on the nervous system. This is unquestionable in four of the cases, and in so far as demonstrating the mode of the action of alcohol on the human lungs is concerned, it is equally true of the other cases; for I think it is pretty well established that phthisis is but the legitimate offspring of any persistent catarrhal state of the lungs, and that chronic bronchitis, and catarrhal and broncho-pneumonia, are but the milestones marking the pathway along which the disease travels to its final destination.

Such then being the relation between alcoholism and pulmonary phthisis it is very readily understood why these two diseases should so frequently change places in different members or generations of the same family, and why they are so often associated with various other nervous disorders. Moreover, alcohol having the potency to produce phthisis *de novo* in the human subject, either directly or through hereditary influence, or both, as we have seen, it must, in view of its past and present widespread abuse in civilized countries,

be a tremendous factor in maintaining the ranks of the hundreds of thousands of those who are annually slain by this terrible malady. To this and to no other conclusion do the premises of this paper point, and if one had the inclination to moralize on this subject it would be very interesting to inquire why the North American Indian, and other savages, were practically free from pulmonary consumption until they came in contact with the white race! When we connect the facts that alcohol and syphilis are the greatest curses which the Indian has acquired from his white civilizer, with the evidence which has been brought forward in this as well as in another paper on *Syphilitic Phthisis*,* I think it must be obvious that these two causes are largely responsible for sowing the seeds of pulmonary phthisis among these people.

* See *The Polyclinic*, February, 1889. In this paper pathological facts and illustrative cases are given to show that syphilis, like alcohol, produces pulmonary consumption by vitiating the nervous system and especially the pneumogastric nerves.